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# Regaining a Perfect Stride

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# Regaining a Perfect Stride

Thoroughbred horse owners recognize the disease known as "wobbles" but very few are bold enough to admit one of their horses has the disease. In the past diagnosis of wobbler syndrome has meant the future for the horse was bleak at best. The disease is particularly prevalent in Thoroughbred horses.

Wobbles is characterized by incoordination, ataxia and leg weakness that usually begins in the hind legs but can progress to involve the forelimbs as well. There are several diseases which can cause "wobbles" in young horses but in each disease the spinal cord in the neck is damaged. The most common causes of spinal cord disease in young Thoroughbred horses are cervical vertebral malformation/malarticulation (CVM); equine degenerative myeloencephalopathy (EDM); equine herpes virus-1 myeloencephalitis; equine protozoal myeloencephalitis (EPM); spinal cord trauma; and, trauma to the vertebrae of the neck. These diseases can occur any time in the life of a horse but are more commonly seen during the first two years. Unfortunately, early subtle signs of these diseases can be overlooked or be initially attributed to clumsiness. Even when dramatic acute signs are observed, diagnosis based on clinical signs and ancillary tests may not be possible.

In spite of the progress made by veterinarians in recent years, improved techniques for diagnosis and treatment of spinal cord disease in young horses are still needed. Early, specific diagnosis and treatment could limit damage to the spinal cord and allow affected horses to recover completely.

At the University of Pennsylvania's School of Veterinary Medicine, we have chosen to concentrate our efforts on methods to improve the diagnosis and treatment of cervical vertebral malformation/malarticulation (CVM). This form of wobbles is caused by direct damage to the spinal cord by the neck bones. When the bones of the neck are malformed or the spinal canal through which the spinal cord passes is too small, the spinal cord can be damaged as the horse moves its neck during normal activities. Severe acute injury to the spinal cord can occur if the neck bones are malformed and the horse moves its neck abruptly or through a greater arc than normal. The damage can be permanent and devastating but more often the early damage to the spinal cord is in the form of inflammation and swelling of the nervous tissue. When the spinal cord is inflamed or damaged, messages from the brain passing to the muscles of the legs are slowed or do not arrive. Untreated, the damage to the spinal cord progresses until the horse has difficulty in walking and becomes a danger to those who must care for it. The damage is very similar to the spinal cord injury a human being can experience by hitting the head and flexing the neck during a dive into shallow water in a swimming pool.

The role of heredity in CVM has been debated for years. CVM has been seen in certain Thoroughbred horse families leading investigators to speculate that there was a genetic trait passed on from one generation to the next. We have observed that the spinal canal in the neck of young horses varies considerably in size. The spinal canal is largest in females. Since 85% of the horses that develop CVM are males, it may be that a sex related characteristic predisposes young male Thoroughbred horses to CVM because they are born with a narrower spinal canal and pinching of the spinal cord can occur more easily.

CVM has also been proposed to be a "developmental orthopedic disease." With this in mind, a relationship between nutrition and the occurrence of CVM has been suggested. It has been found that horses with CVM have often been overfed relative to other horses. This in turn causes rapid growth. It has been well demonstrated that overfeeding of a high protein, high energy diet to young growing animals and children can cause bony

**Dr. Donawick examining a horse for Wobbles.**



deformities. For instance, great Danes developed spinal cord compression when overfed from birth. On the basis of numerous indications that over nutrition is associated with the development of orthopedic diseases, such as CVM in horses, we undertook a study to determine if CVM could be diagnosed early in life in Thoroughbred foals and, if CVM was found, we wanted to see if dietary modification along with confinement could eliminate the neurologic signs and correct the bony abnormalities.

Our work has been conducted on a Thoroughbred breeding farm in central Kentucky, between 1986 and 1990. The international research team includes Dr. Ian (Joe) Mayhew, a world renowned veterinary neurologist from Newmarket, England; Dr. Sherril Green, from the Ontario Veterinary College at the University of Guelph in Canada; Dr. David Galligan, a veterinary nutritionist at the University of Pennsylvania; Liza Stanley, a graduate student; and myself. The farm experienced a high incidence of CVM before and during the study. During the five years 132 foals were born to 43 mares on the farm. Eighteen of the foals developed CVM.

To diagnose the condition early, often even before neurologic signs developed, we took radiographs of the necks of the young horses. The neck bones were examined for such things as narrow spinal canal, abnormal growth of the joints connecting the neck bones, abnormal growth plates at the ends of the neck bones and enlarged bony roofs over the spinal canal. The changes were graded for each of the seven bones of the neck and the radiographs on each foal were given an overall CVM score. The scale was zero (perfect) to 35. Through experience, we found that when the overall CVM score was greater than 12 there was a very high probability that the young horse had or would soon develop neurologic signs of CVM. Hundreds of radiographs have been scored and the results have been entered into a computer so that when new horses are examined they can be compared with horses seen in the past. In this way, the accuracy of our diagnostic methods has continued to improve with time. We can now examine a set of neck radiographs taken with the young horse standing, not under anesthesia, and based on the radiographic score, predict whether there is cause for concern.

All of the foals, weanlings and yearlings, on the Kentucky farm were examined every three to four months to see if they had developed any signs of neurologic disease. In addition, they were weighed on a weekly basis to monitor their growth rates.

If a young horse developed clinical signs of spinal disease and it had a high overall CVM score in its neck radiographs it was treated in two ways. First, the young horse was confined to a stall to limit neck

movement and activity. Secondly, it was placed on a restricted diet to slow growth and offer time for bone remodeling. The young horses were fed between 65 and 75% of the normally recommended amounts of protein and energy. Special attention was taken to insure all of the necessary vitamins and minerals were given and to be sure that the diet was balanced in all other ways.

We were gratified when we saw the first of the treated horses recovered completely. We have been astonished to see that all 18 of the treated horses have recovered completely. It has taken about 9 months of treatment before the improvement in the neck has been sufficient to permit discontinuation of the diet and confinement. It is important to note that all of the treated animals were less than one year of age when treatment was started. We believe this fact is very important. Success is undoubtedly dependent on starting treatment while bone growth is most rapid.

All 18 of the treated horses have entered race training. Of the ten horses treated between 1986 and 1988, which are now old enough to race, seven have raced, and 5 have won at least one race. In actuality, the 5 horses that have raced have won a total of 14 races!

We have expanded our studies to include young horses at other farms throughout the United States and Canada. Working closely with local veterinarians and owners it has been possible to achieve gratifying results in these less well controlled surroundings, as long as the owners, trainers and veterinarians adhere to the program and are understanding. It is hard for owners to see their animals confined for long periods and to be unthrifty. There is the temptation to want to add food or turn the young horse out for exercise, both of which can undo all that has been accomplished. From experience we know the horses will grow and reach their predestined potential, but it is hard to convince others in the midst of treatment.

The future holds great promise. The accuracy of our ability to diagnose CVM will undoubtedly improve as our base of information increases. Further study will enable us to determine what specific dietary changes are making the most impact in treatment of the disease. The knowledge gained during our studies of the horse may have broad application to other species including man. For the Thoroughbred industry, the role of diet for the young horse must be underscored as breeders think towards the future and their chances for success of consistently making it to the winners circle.

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